REVIEW ARTICLE

Observations on the relation between alcohol absorption and the rate of gastric emptying

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Alcohol (ethanol) is absorbed slowly from the stomach and rapidly from the small intestine, and the rate of its absorption depends on the rate of gastric emptying. When gastric emptying is fast, the absorption of alcohol is fast. When gastric emptying is slow the absorption of alcohol is delayed and peak blood alcohol concentrations are reduced. Alterations of the gastric emptying rate, which may have a physiologic, pharmacologic or pathologic cause, markedly influence the rate of alcohol absorption. The gastric emptying rate makes an important contribution to inter- and intraindividual variations in the rate of alcohol absorption and therefore the timing and magnitude of the acute intoxicating effect of an oral dose of alcohol.

L'alcool (éthylique) est absorbé lentement dans l'estomac et rapidement dans le petit intestin, et la vitesse d'absorption de l'alcool dépend du taux de vidange gastrique. Quand la vidange gastrique est rapide, l'absorption de l'alcool est rapide. Quand la vidange gastrique est lente, l'absorption de l'alcool est retardée et les concentrations sanguines maximums en alcool sont diminuées. Des changements du taux de vidange gastrique pouvant avoir des causes physiologiques, pharmacologiques ou pathologiques, influencent profondément le taux d'absorption de l'alcool. Le taux de vidange gastrique apporte une importante contribution aux variations observées entre les individus et chez un même individu dans la vitesse d'absorption de l'alcool et, en conséquence, sur le moment et l'importance des effets d'une intoxication aiguë par une dose orale d'alcool.

Although alcohol is a readily diffusible compound of low molecular weight it is absorbed much more slowly from the stomach than the small intestine.[1,2] The small bowel is the most important site for the absorption of all drugs, mainly because the large surface area presented by the villi of the small intestine has a greater influence than other factors that determine drug absorption.[3,4] Normally the amount of alcohol absorbed from the stomach is small compared with the amount absorbed from the small bowel.[2] The introduction of 200 ml of 37% ethanol into the stomach of a dog, with the pyloric opening of the stomach ligated, may be followed by almost complete disappearance of the alcohol from the stomach lumen in 3 hours.[2] However, when the pylorus is patent the rate of alcohol absorption is greatly increased because alcohol gains access to its principal site of absorption, the small intestine.[2]

There is much variation from one person to another in the timing and magnitude of the intoxicating effect following ingestion of a given quantity of alcohol. This variation has been largely explained in terms of interindividual differences in the complex process of the development of tolerance, especially in relation to alterations in the rate of metabolism of ethanol.[5,6] The fact that this variable response could be related in part to alterations in the rate of gastric emptying, and therefore to altered rates of alcohol absorption, has received much less attention. Few factors can substantially alter the rate of alcohol metabolism enough that major alterations in the blood alcohol concentration are noted after oral alcohol intake, but many factors can alter the rate of absorption of alcohol and other drugs.[7,8] Since the rate of catabolism of alcohol is slower than the rate of absorption, metabolic tolerance will not substantially alter the peak effect achieved with acute alcohol intake.[8] Therefore, the speed with which alcohol is absorbed and distributed to the central nervous system, where it exerts an intoxicating effect, may play a major role in determining the intoxicating effect of a given dose of alcohol. Since the rate of transfer of alcohol from the stomach to the small bowel markedly influences the rate at which alcohol is absorbed, the motility of the stomach is often a major rate-limiting factor in alcohol absorption.

Although there are a number of excellent reviews of the physiologic aspects of alcohol absorption in the literature,[9-17] no recent article has examined this topic with a clinical perspective. The objects of this paper are to draw attention to the im-

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portance of the gastric emptying rate as a determinant of the alcohol absorption rate and to review how certain factors, including drugs, diseases and alcohol itself, may alter gastric motility, thereby influencing the rate of alcohol absorption.

How gastric emptying and alcohol absorption are related

Much interest has been directed to the relation between the gastric emptying rate and the drug absorption rate.[3,4,7,18-20] Ethanol and weakly acidic drugs, including acetaminophen, warfarin, barbiturates and salicylates, are all absorbed more rapidly from the small bowel than from the stomach. The rate of acetaminophen absorption is directly related to the rate of gastric emptying;[20,21] this finding has led to the use of the absorption kinetics of orally administered acetaminophen and other drugs, such as alcohol, as an indirect assessment of the gastric emptying rate in humans.[19]

The knowledge that alcohol absorption occurs principally from the small bowel[17,22,23] led to the hypothesis that there may be a direct relation between the alcohol absorption rate and the gastric emptying rate in humans.[24-28] On the basis of this assumption Finch and colleagues[24] in 1974 suggested using a breath alcohol analyser after the ingestion of a standard quantity of alcohol to assess the gastric emptying rate in humans. They reasoned that altered rates of delivery of alcohol from the stomach to the small bowel should be reflected in altered rates of alcohol absorption. Since alcohol is excreted by the lungs, and its concentration in expired air is directly proportional to the pulmonary capillary blood concentration, a breathalyser can be used to measure blood alcohol levels,[29-31] with certain limitations.[32] Using a breath alcohol analysis instrument to estimate blood alcohol concentrations Finch and colleagues produced reproducibly smooth blood ethanol curves, with about 10% intrasubject variation in peak concentrations. The blood alcohol concentration–time curves, as determined by the breathalyser, were modified by the administration of metoclopramide hydrochloride to increase the motility of the upper gastrointestinal tract and propantheline bromide to decrease the motility. An increase in motility resulted in a rapid rise in blood alcohol levels, with higher peak concentrations, and a decrease in motility resulted in flattened absorption curves compared with control curves.

Since the available methods of measuring the gastric emptying rate in humans have disadvantages or limitations, such as gastric intubation or exposure of the patient to ionizing radiation,[33,34] any simple noninvasive test of gastric emptying would be of obvious value. To validate the use of the alcohol absorption rate as an indicator of the gastric emptying rate in humans my colleagues and I undertook a study to examine directly the relation between gastric emptying and alcohol absorption.[35] Alcohol absorption, as determined by simultaneous measurements of breath and blood alcohol concentrations, was measured at the same time as the gastric emptying rate in eight male volunteers. After the subjects had ingested absolute alcohol (0.5 g/kg of body weight) diluted in 500 ml of orange juice, their gastric emptying rates were measured by sequential scintiscanning.[20,21,36] We found a significant positive correlation between alcohol absorption (as assessed from the area under the venous alcohol concentration–time curve at 30 and 60 minutes) and gastric emptying (as assessed from the percentage of the ingested solution emptied from the stomach at 30 and 60 minutes). The demonstration of a direct dependence between the rate of alcohol absorption and the rate of gastric emptying supports the suggestion that the rate of absorption of alcohol, under the conditions of this study, may be useful as an indirect assessment of the gastric emptying rate in humans.

The breathalyser may be sufficient in certain circumstances to detect gross alterations in gastric emptying, but there are a number of limitations to its use.[28,35] For example, the early phases of gastric emptying and alcohol absorption cannot be assessed because the breath alcohol readings may be spuriously high owing to contamination of the oropharynx by the ingested alcohol[37] even after the mouth has been rinsed with water.[35] In addition, the results of a breathalyser study may be influenced by the accuracy of the breath alcohol analysis instrument, errors in instrument calibration, the temperature and humidity of the environment, pathologic conditions of the respiratory tract and even whether the subject had his or her mouth open or was talking before breath sampling.[38] Furthermore, the blood alcohol concentration–time curve is determined by a variety of factors,[17] including the rate of metabolism and distribution of alcohol, and some alcohol may be absorbed in a nonlinear manner from the stomach bed.[39,40] These circumstances, together with the fact that alcohol itself may exert an effect on gastric emptying, severely limit the accuracy of gastric emptying rates determined by means of a breathalyser. Thus, it appears that a breathalyser can be used only as a relatively crude index of the gastric emptying time.

Matilla and associates,[28] studying the effect of aluminum hydroxide and glycopyrronium bromide on the absorption of ethambutol and alcohol, were unsuccessful in using repeated breath alcohol analyses to assess the time course of gastric emptying. They suggested that the lack of an abrupt peak in the breath alcohol concentration indicated that the method was not detecting pyloric opening and was therefore not useful for their purpose.

Physiologic influences on alcohol absorption

Since the alcohol absorption rate is dependent upon the gastric emptying rate, any factors that alter gastric motility will therefore alter the alcohol absorption rate. Although changes in the gastric emptying rate alter the rate of alcohol absorption they do not usually alter the total amount of alcohol absorbed from the gastrointestinal tract. Gastric emptying may account for much of the observed variation in the alcohol absorption rate since gastric emptying is influenced by many factors,[41,42] including food and its energy density,[43,44] the volume,[45] pH,[46] composition,[47] viscosity[20] and osmolarity of the gastric contents,[45] autonomic and hormonal activity,[45] emotional state,[42] pain,[48] posture,[49] surface active agents,[50] cigarette smoking[51] and many commonly used drugs,[7] including ethanol.[52]

The presence of food in the stomach, the type of alcoholic beverage and individual constitutional factors all influence the speed with which alcohol leaves the stomach and therefore the rate of alcohol absorption.[7,17,53] A large intersubject variation in the alcohol absorption rate was reported by Dubowski[54] in 1976 from a pharmacokinetic study of orally administered ethanol in 41 healthy volunteers. Dubowski found that the interval from the end of alcohol intake to the time of peak blood alcohol concentrations in his subjects was highly variable, with a range of 14 to 138 minutes in the men (mean ± standard deviation [SD] 56.7 ± 36.5 minutes; coefficient of variation 64.4%) and 15 to 84 minutes in the women (mean ± SD 41.9 ± 22.1 minutes; coefficient of variation 52.7%). The volunteers had consumed known amounts of ethanol ranging from 0.43 to 1.6 g/kg, in a dilution and at a rate preferred by each, together with a sandwich. Since altered gastric emptying exerts a major influence on the interval between oral administration of a drug or alcohol and the time of peak blood concentrations, it seems likely that variations in the gastric emptying rate, in addition to differences in the alcohol consumption rate, accounted for much of the variation in the alcohol absorption rate.

Many studies have demonstrated that when alcohol is ingested together with or after food it is absorbed more slowly than when it is consumed on an empty stomach.[55-62] Although the main mechanism for slower absorption of alcohol with food is a delay in gastric emptying, dilution of ethanol by food or fluid in the gastric lumen may play a role.[17] Lin and coworkers,[61] studying the entire time course of blood alcohol concentrations following ingestion, found that the timing and level of the peak alcohol concentration and the area under the blood alcohol concentration–time curve were markedly reduced when alcohol was ingested along with solid food rather than alone.

A delay in alcohol absorption is noted when alcohol is ingested along with most foods, especially fatty or heavy, solid, proteinaceous meals, which are known to retard the gastric emptying rate.[44,63-65] However, there have been conflicting opinions concerning the effect of fat on alcohol absorption. Some researchers have indicated that olive oil may actually increase the rate of alcohol absorption,[53] while others have reported that olive oil delays alcohol absorption.[66,67] These different observations may be due to a variable effect of fat on gastric motor function: although fat ingested alone or in test meals normally slows gastric emptying, it may on occasion induce nausea, which could, in the absence of pylorospasm, accelerate gastric emptying and alcohol absorption.[17] There appears to be a wide interindividual variation in the degree to which fat-containing products such as milk delay the absorption of alcohol, thereby protecting against high blood alcohol levels.[58] Hence, the common practice of taking milk before ingesting alcohol to reduce the level of inebriation[58] may not always prove successful unless, perhaps, a large quantity of milk or of concentrated milk products is taken. In one study 240 ml of milk taken on an empty stomach shortly before alcohol ingestion afforded only marginal protection against high blood alcohol levels.[58]

The type and therefore the alcohol content of the beverage consumed influence the alcohol absorption rate:[17] wine is absorbed faster than gin and whisky, but not as fast as pure ethanol.[17,68] It has been suggested that congeners in alcoholic drinks may affect absorption, possibly by altering the gastric emptying rate.[68] Other components of alcoholic beverages may be important in determining the alcohol absorption rate: bicarbonate in sparkling wine and "mixes" taken with spirits accelerate absorption, whereas sugars in sweet drinks retard absorption.[17] This occurs because carbonated liquids increase but sugars reduce the gastric emptying rate. Food and alcohol modify the blood flow in the gastrointestinal mucosa[69] and the mesenteric lymph flow,[70] but these factors generally have less influence on alcohol absorption than altered motility of the upper gastrointestinal tract.

Not only is alcohol absorbed very quickly from the small bowel, but also it can rapidly diffuse through the stomach wall. In the first 20 minutes after ingestion, up to 43% of an administered dose of alcohol may be absorbed through the stomach wall.[39] The overall absorption of ethanol appears to be greatest when the ingested concentration is between 15% and 30%;[17] in animals and humans if the concentration is greater than 30% pylorospasm may occur, with a resultant delay in alcohol absorption.[53] Pylorospasm and subsequent vomiting after alcohol intake may be more likely in occasional drinkers than in heavy drinkers.[53] The pylorospasm appears to have a protective function, as the closing of the gastric outlet is associated with vomiting or delayed absorption, which will limit the overall toxic effect of the ingested alcohol.

A variety of complex, interrelated factors may alter the gastric absorption of alcohol. Davenport[71] has indicated that the amount of alcohol absorbed from the stomach is directly related to its gastric luminal concentration. This concentration is influenced by factors such as the amount or concentration of alcohol ingested,[72] whether food is ingested at the same time[40] and whether gastric acid secretion is inhibited[73] or stimulated[74] by ethanol. Alcohol in high concentrations may result in marked functional and structural alteration in the gastric mucosa,[71] manifested by mucosal erosions and hemorrhages,[75,76] interference with smooth muscle contraction[77] and increased blood flow in the gastric mucosa.[69] All of these circumstances will influence absorption from the stomach and alter the transfer of alcohol to its principal site of absorption, the proximal small bowel.

Alcohol has a variable effect on gastric emptying that appears to be related largely to the concentration of alcohol in the ingested fluid.[68] Experimental observations in animals[78] and humans[52] have confirmed the dose-dependent effect of alcohol on gastric evacuation. In low concentrations (about 6 g/dl) alcohol does not affect the gastric emptying rate,[79] but solutions of ethanol may empty more rapidly than glucose solutions of similar osmolarity.[80] Recently Kaufman and Kaye,[81] using alcohol-containing meals of equivalent energy value, have shown that the duodenal osmoreceptor mechanisms that control gastric motility are insensitive to alcohol and that the relation between the energy density of food and the gastric emptying rate does not exist for ethanol.[44] This is to be expected since ethanol diffuses readily across the intestinal mucosa and therefore may not exert any osmotic pressure on the intestinal mucosal membrane.[82] A balance between altered gastric emptying and an increased alcohol concentration explains the relation between an increased alcohol concentration and the altered absorption rate.[17]

Drugs, diseases and alcohol absorption

Metoclopramide,[25] cholinergic agents,[22] chlorpromazine[83] and insulin-induced hypoglycemia[84] can all increase the alcohol absorption rate; in most instances the increase is due to more rapid gastric emptying. In contrast, alcohol absorption is retarded by agents that delay gastric emptying, including anticholinergic drugs,[25,26] such as atropine, propantheline bromide and tricyclic antidepressants,[26] and by antacids[85,86] and caffeine.[87] In addition, substances that damage the gastric mucosa, such as acetylsalicylic acid and aminopyrine, delay alcohol absorption, probably by inducing a degree of pylorospasm.[68]

Rapid gastric emptying and therefore rapid alcohol absorption are frequent sequels to gastric surgery. The normal gastric emptying pattern is disorganized by vagotomy,[88] and surgical procedures to facilitate stomach drainage, such as pyloroplasty and gastroenterostomy, may result in very rapid transfer of gastric contents into the duodenum.[89,90] Gastric resection is more likely to produce "gastric incontinence" than more recent operations for peptic ulcer, such as highly selective vagotomy.[91,92] A number of workers have investigated the effect of gastric surgery on a patient's "tolerance" to a given oral dose of alcohol.[92-95] In general, the operations that tend to be followed by increased blood alcohol concentrations and intoxication are those that lead to rapid transit of gastric contents into the small bowel. Some authors have drawn attention to the importance of warning patients of an increased susceptibility to the inebriant effect of alcohol after they have undergone gastric surgery.[93,94] Furthermore, detailed studies have indicated that short-term memory,[96] cognitive performance[97] and simple reaction time[98] tend to be impaired more on the ascending limb than on the descending limb of the blood alcohol curve. In the case of reaction time it has been hypothesized that "adaptation" may account for this finding.[98] Memory impairment may be related to a sudden rise in the blood alcohol level,[96] a rise that tends to be more rapid following gastric surgery and allows less time for "adaptation". These findings reinforce the need to warn patients who may absorb alcohol rapidly, and they may be especially important in relation to road traffic legislation.[93]

There have been few studies of the effect of gastrointestinal and systemic diseases on alcohol absorption. It has been suggested that nausea may on occasion lead to an acceleration of gastric emptying and alcohol absorption, whereas intense mental effort and physical exertion tend to slow alcohol absorption.[99,100] Jaulmes and colleagues[101] in 1956 reported that a reduction of body temperature may retard alcohol absorption, whereas an increase in body temperature accelerates absorption.[102] In patients with long-standing insulin-dependent diabetes who have autonomic neuropathy, gastric emptying may slow;[103] this is in accord with the low, flat blood alcohol curves observed in some diabetic patients.[104] However, insulin-induced hypoglycemia accelerates gastric emptying and alcohol absorption,[84] and this could contribute to an adverse interaction between insulin and alcohol in a diabetic individual since both substances can induce hypoglycemia.

In persons with certain diseases, variables other than gastric emptying, such as metabolic rate disturbances and gastrointestinal absorptive defects, could potentially influence both the alcohol absorption rate and "tolerance" to alcohol. In one study, survivors of concentration camps with severe malnutrition appeared to absorb alcohol rapidly.[105] This finding is difficult to explain in terms of the gastric emptying rate since gastrointestinal motility seems to be generally reduced in malnourished individuals,[106] and a delay in gastric emptying has been noted in healthy volunteers during starvation.[107] Similar difficulty surrounds the explanation of the altered alcohol absorption observed in hyperthyroid animals and humans. Alcohol was absorbed from the gut more rapidly in rats rendered hyperthyroid by treatment with 3,3′,5-triiodo-L-thyronine than in normal or propylthiouracil-treated hypothyroid animals.[108] Acetaminophen absorption and, by inference, gastric emptying[20,21] are more rapid in humans with hyperthyroidism,[109] but in limited studies of alcohol absorption in thyrotoxic patients the blood alcohol concentration–time curves were seen to be slightly lower and flatter.[104] This finding may illustrate a potential role for other factors, perhaps metabolic phenomena, as determinants of blood alcohol levels following alcohol ingestion. Patients with celiac disease have flatter alcohol absorption curves[94] and slower acetaminophen absorption (with reduced and delayed mean and peak plasma acetaminophen concentrations)[110] than healthy subjects. Simultaneous measurements of the gastric emptying rate and acetaminophen absorption together with compartmental pharmacokinetic analysis have indicated that the slower absorption of acetaminophen in these patients is due to delayed gastric emptying of liquids.[110] Despite an initial impression of drug malabsorption from consideration of the early phases of the plasma acetaminophen concentration–time curves, we found that the urinary recovery of the drug was similar in the patients and the controls; drug malabsorption did not occur. Therefore, it is likely that the flatter alcohol absorption curves observed in patients with celiac disease represent slower alcohol absorption, which is probably due to a delay in gastric emptying of liquids rather than malabsorption of alcohol.

Comments

The rate of alcohol absorption has an important influence on the degree of alcohol intoxication that occurs with acute alcohol intake.[111] The more rapid the rate of absorption, the more intoxicated an individual may become.[8,97,111] Since the rate of alcohol absorption is dependent on the rate of gastric emptying, changes in gastric motility are an important factor in the degree of inebriation reached following the ingestion of alcohol. The alcohol in distilled liquors is absorbed faster, because of more rapid gastric emptying, than the alcohol in beer, and a single large dose of liquor will produce greater intoxication than an equal dose of alcohol contained in beer. This helps explain the basis of the many references in folklore and nonscientific literature to the specificity of the effect of different alcoholic drinks.[111] The awareness that drinking alcoholic beverages that are rapidly absorbed will cause greater inebriation could play an important role in drink selection and perhaps change drinking habits. Alcoholics may start a drinking episode by rapidly ingesting large volumes of alcohol to achieve quickly peak blood alcohol levels and therefore greater intoxication.[8] However, it should be emphasized that alterations of the gastric emptying rate as a major determinant of the alcohol absorption rate are most important in acute alcohol intake.

Although this paper has focused on biomedical factors, especially the gastric emptying rate, that influence alcohol absorption and therefore intoxication, the potential importance of psychologic variables as determinants of the response to alcohol should be considered. For example, at low to moderate blood alcohol concentrations an individual's expectation about the effects of what he or she is drinking may, on occasion, override the pharmacologic action of alcohol. Experiments have shown that, despite the actual content of drinks (e.g., vodka and tonic v. tonic only), individuals who perceive that they have consumed alcohol tended to exhibit less social anxiety,[112] greater sexual arousal[113] and more aggressive interpersonal behaviour.[114] Thus, a more complete understanding and prediction of overt responses to alcohol intake must consider both biomedical and behavioural factors. Alcohol preference and consumption are regulated by a complex interaction of social, psychologic and biologic variables. However, some variables are easier to alter than others, and it is clear that the absorption rate is easier to change than many other variables.[115]

A knowledge of the interactions between the gastric emptying rate and the alcohol absorption rate helps one understand the circumstances that determine the degree of intoxication resulting from acute alcohol intake. Because of the variability in the alcohol absorption rate it is hazardous to predict the peak blood alcohol concentrations during moderate or excessive drinking. And since this rate influences greatly the shape of the blood alcohol concentration–time curve, acceptably precise predictions of blood alcohol concentrations such as may be requested in forensic practice are impossible.[54]

Conclusion

Alcohol, like many compounds, is absorbed more rapidly from the small intestine than from the stomach. The gastric emptying rate is an important determinant of the alcohol absorption rate, and differences in the absorption rate may often be clearly related to differences in gastric emptying. An increasing awareness of the many factors that control gastric emptying and therefore influence alcohol absorption has contributed to our understanding of the causes and significance of the inter- and intraindividual variations in the rate of absorption of alcohol.

I thank Professor Y. Israel, Professor H. Orrego and Dr. H.A. Skinner for helpful advice during the preparation of this manuscript. Support from the Addiction Research Foundation and the Carnegie Trust for the University of Scotland is greatly appreciated.

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hypertensive encephalopathy. Intravenous pyelograms and computed tomograms were normal.

Oral therapy with hydralazine hydrochloride and hydrochlorothiazide was started, and the patient's blood pressure stabilized at 120/85 mm Hg. On the 13th postoperative day lengthening of the right femur was resumed. Two days later the femoral lengthening had reached almost 4 cm and the patient's blood pressure again rose, to 150/110 mm Hg. Oral propranolol therapy was started and her blood pressure fell to 110/60 mm Hg within 24 hours.

She remained normotensive and the lengthening continued to 4.3 cm. On the 64th postoperative day the distraction apparatus was removed and a spica cast applied to the hip under general anesthesia. The procedure was well tolerated and the blood pressure remained normal. The antihypertensive therapy was tapered and finally discontinued 3 months after the patient's discharge from hospital. She continued taking phenobarbital for 1 year.

Three years after the operation the girl had a blood pressure of 110/65 mm Hg and showed no evidence of seizure activity or neurologic deficit. Serial EEGs had revealed improvement, though there was residual irregularity, of unknown significance, in the left posterior temporal region.

Discussion

In 1967 Yosipovitch and Palti[6] suggested that hypertension secondary to limb lengthening procedures is due to increased tension within the sciatic nerve, which results in reflex peripheral vasoconstriction. They postulated that the reflex is independent of cardiac output or adrenal secretion and consists of an afferent limb (the proximal portion of the sciatic nerve), spinal and medullary vasomotor centres, and efferent vasoconstrictor and vasodilator fibres. Adrenergic blocking agents would therefore control the rise in blood pressure.

More recently, Whitehill and Hakala[7] have shown that stretching of *all* the major nerves in the leg is responsible for the rise in blood pressure. Further, they found that, in the dog, drugs that cause α-adrenergic blockade (phenoxybenzamine hydrochloride), catecholamine depletion (reserpine) or ganglionic blockade (trimethaphan camsylate) reduce the blood pressure in this situation. A β-adrenergic blocker (propranolol) has no effect.

Our patient further illustrates one of the many complications of femoral lengthening procedures. The case is unusual in that the seizure, which may have heralded the onset of the hypertension, was late in onset. The patient remained asymptomatic until the 11th postoperative day, when she had convulsions secondary to the hypertension. She did not exhibit the previously reported prodromal symptoms — headache and dizziness. While initially the blood pressure was reduced with antihypertensive therapy, it rose subsequently. Intravenous antihypertensive therapy again reduced the pressure, and at the same time the distraction apparatus was released. However, the blood pressure did not remain low, as it has usually been reported to do after release of this apparatus — another unusual feature of the case. Further lengthening occurred only after the administration of a β-adrenergic blocker. No further problem was encountered.

This case emphasizes the importance of careful routine monitoring of the blood pressure after the operation, as well as prompt release of the distraction apparatus if hypertension occurs. It also shows that β-adrenergic blocking agents are valuable adjuncts to the standard diuretic and antihypertensive medications, despite experimental evidence to the contrary.

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